AMENDMENTS

IN THE SPECIFICATION

At page 1 of the specification after the title please delete current text and add:

--This application is a continuation of copending Application No. 10/051,662, filed January 18, 2002; which is a continuation of Application No. 09/479,837, filed January 7, 2000 (now Patent No. 6,407,082); which is a continuation of Application No. ~~08/073,010~~ 08/873,010, filed June 11, 1997 (now Patent No. 6,034,074)~~; which is a continuation in part of 08/713,834, filed September 13, 1996 (now U.S. Patent No. 6,028,064)~~.--

At page 4, line 7 to page 5, line 13, revise and replace the paragraph by changing two occurrences of "mg" to --mcg-- as shown below:

--Vitamin D deficiency in childhood produces rickets, which is characterized by inadequate calcification of cartilage and bone. In adults, Vitamin D deficiency leads to softening and weakening of bones, known as osteomalacia. The major therapeutic uses of Vitamin D are divided into four categories: (1) prophylaxis and cure of nutritional rickets, (2) treatment of metabolic rickets and osteomalacia, particularly in the setting of chronic renal failure, (3) treatment of hypoparathyroidism, and (4) prevention and treatment of osteoporosis. Recommended daily dietary allowances of Vitamin D by the Food and Nutrition Board of the United states National Research Council (1989) were 10 mcg cholecalciferol (400 IU Vitamin D) daily for females age 11-24 and 5 mcg cholecalciferol (200 IU Vitamin D) daily for females age 25 and older. Normal serum levels of 25-hydroxyvitamin D.sub.3 are not closely regulated and it has a biological half-life of several weeks with blood levels typically ranging from 15 to 80

ng/mL. Serum levels of 1,25dihydroxyvitamin D.sub.3 are more closely regulated and typically range from 15-60 pg/mL. Serum 1,25-dihydroxyvitamin D.sub.3 has a half-life of 6-8 hours. 1,25-dihydroxyvitamin D.sub.3 partitions into cells by virtue of its lipophilicity, binds to intracellular receptors, and translocates to the nucleus where the complex controls the transcription of a number of genes, many of which relate to calcium metabolism. Corder et al., Cancer Epidemiology, Biomarkers & Prevention 2:467-472 (1993).--

At page 15, line 30 to page 16, line 18, revise and replace the paragraph by changing four occurrences of "mg" to --mcg-- as shown below:

--Appropriate dosages to increase the induction of apoptosis of non-neoplastic ovarian epithelial cells may be determined by those of skill in the art depending upon the identity of the Vitamin D compound and its method of administration. For example, preferred dosages of the Vitamin D compound effective to increase apoptosis of non-neoplastic ovarial epithelial cells range from 0.0001 to 1.0 mcg/kg of body weight (based upon the apoptotic potency of 1,25-dihydroxyvitamin D.sub.3) with dosages ranging from about 0.005 to 0.75 mcg/kg being more preferred and dosages of about 0.05 to 0.5 mcg/kg being particularly preferred. It is hypothesized that even higher dosages of 1,25-dihydroxyvitamin D.sub.3 may be more effective in inducing apoptosis. A Vitamin D analogue that has greater potency than 1,25-dihydroxyvitamin D.sub.3 in inducing apoptosis and/or which does not have the deleterious side effects of 1,25-dihydroxyvitamin D.sub.3, such as hypercalcemia, could be administered at a dosage equivalent much higher than 1.0 mcg/kg of 1,25-dihydroxyvitamin D.sub.3. While the potency and bioavailability of other Vitamin D compounds and analogues may vary, those of skill in the art

can determine their apoptotic potency in relation to 1,25-dihydroxyvitamin D.sub.3 and appropriate dosages and regimens of administration through use of in vitro testing methods such as disclosed in the accompanying example. --

At page 18, line 13 to page 19, line 11, revise and replace the paragraph by changing two occurrences of "mg" to --mcg-- as shown below:

--The term "progestin product" as used herein includes any drug which binds to the progestin receptor and induces a progestational effect. This definition thus includes all of the known progestins, derivatives of progesterone or testosterone that have progestin activity, and progestin agonists. It is contemplated that not only presently available progestins but also progestins introduced in the future will be useful according to the present invention. The known synthetic progestins are mainly derivatives of 17-alpha-hydroxy-progesterone or 19-nortestosterone. These progestins can be classified into three groups: the pregnane, estrane, and gonane derivatives. Progestin products may be administered at a variety of dosages including at a dose less than or equal to a dose equivalent to 10 mg daily of norethindrone, more preferably less than or equal to 1 mg daily, or less than or equal to 0.2 mg daily, and possibly as low as 0.05 mg daily of a norethindrone equivalent dose. According to a preferred aspect of the invention, a vitamin D compound and a progestin may be coadministered as a pharmaceutical composition preferably in a single unit dosage, such as a tablet, for inhibiting the conversion of non-neoplastic ovarian epithelial cells to neoplastic cells. The pharmaceutical composition comprises a Vitamin D compound and a progestin product in amounts which are together effective to increase apoptosis in non-neoplastic ovarian epithelial cells. Preferred pharmaceutical

compositions include those wherein the Vitamin D compound is present at a dosage equivalent of from 0.0001 to 1.0 mcg 1,25-dihydroxyvitamin D.sub.3/kg of body weight and wherein the progestin product is present at a dosage less than, or equal to, a dosage equivalent to 10 mg of norethindrone or 1 mg of norethindrone. More preferred compositions comprise those wherein the Vitamin D compound is present at a dosage equivalent of from 0.005 to 0.1 mcg 1,25-dihydroxyvitamin D.sub.3/kg of body weight and wherein the progestin product is present at a dosage less than or equal to a dosage equivalent to 1 mg of norethindrone.